with bicuspid aortic valves excluded from the study? Did patients with Gothic arches and bicuspid valves have even greater changes? Did any of the patients with Gothic arch and a tricuspid aortic valve have aortic insufficiency or require valve replacement/repair?

Approximately 15% of patients who are status-post repair of type I aortic dissection require replacement of the proximal descending aortic owing to increasing diameter on long-term follow-up. It has also been my impression that those patients who have repair of type I aortic dissection with replacement of the ascending aorta and hemiarch repair have a greater incidence of progressive aortic insufficiency and may require aortic valve replacement or aortic root replacement. This cohort is in contrast to those patients with type I aortic dissection who simply had ascending aortic replacement and appeared to have a lesser incidence of aortic insufficiency and valve or root replacement over the long term.

Might the hemodynamic changes that were documented in the pediatric angular Gothic arch be present in this status-post aortic dissection group and predispose to aortic valve insufficiency and dilation of the proximal portion of the distal descending thoracic aorta? Might this effect be more pronounced in the hemiarch repair that predisposes to an angular Gothic arch configuration? Replacement of the ascending aorta only (without hemiarch repair) usually preserves the concave configuration of the ascending aorta and its gentle curve into the transverse arch. Ascending aorta and hemiarch replacement typically has an angulated Gothic configuration rather than the gentle curve that nature favors.

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Aprotinin and renal dysfunction: The role of exposure to angiotensin-converting enzyme inhibitors

To the Editor:

I read with great interest the recent article by Pagano and colleagues[1] detailing their favorable experience with aprotinin in a large patient series (N = 7836: 1998–2006) from a single institution. The authors demonstrated that aprotinin exposure did not significantly affect the incidence of postoperative renal dysfunction.

Careful review of this interesting paper reveals, however, that although hypertension was significantly more common in the aprotinin cohort (63.3% vs 55.1%; $P < .001$), the incidence of exposure to angiotensin-converting enzyme (ACE) inhibitors is not indicated. This is an important confounder, inasmuch as ACE inhibitors in conjunction with aprotinin have been shown to be associated with renal dysfunction after cardiac surgery.[2,3]

As a consequence of this observation, I have the following questions:

1. What was the incidence of ACE inhibitor therapy in the aprotinin cohort?
2. Was exposure to ACE inhibitors significantly different in the aprotinin cohort?
3. What was the percentage of off-pump coronary bypass procedures, given that aprotinin and ACE inhibitors have recently been significantly associated with postoperative renal dysfunction in this patient subgroup?[3]
4. Could the incidence of ACE inhibitor exposure have confounded the results of the study, given the findings from the literature?[2,3] For example, could a lack of renal toxicity from aprotinin be explained by a low incidence of exposure to ACE inhibitors in the aprotinin cohort?

I congratulate the authors again on their important contribution. I look forward to their feedback about the impact of ACE exposure and aprotinin on renal dysfunction after cardiac surgery.

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Reply to the Editor:

Dr Augoustides raises an appropriate point. It is the policy in our unit to discontinue angiotensin-converting enzyme inhibitors or equivalents 24 hours before the operation and for patients with impaired renal function, at least 48 hours before the operation. However our database does not have information on compliance to this policy. Therefore, we could not address this issue in our article.[1]

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Prosthetic valve thrombosis: A regimen of treatment with low-dose and longer-course using recombinant tissue-type plasminogen activator is a promising protocol

To the Editor:

Despite the progress in anesthesia, cardiac surgery, and perioperative care, the therapeutic decision in prosthetic valve thrombosis (PVT) remains in discussion.

In recent years thrombolytic therapy has won acceptance, and for many it is the first

therapeutic choice because the mortality is lower than that of surgical treatment and its application is easy and rapid.[1,2]

We do not know with certainty how long it takes thrombolytic therapy to deocclude a thrombosed prosthesis, although it probably takes less time than surgery because of all the equipment needed to implement aggressive treatment.

The great risk of a redo valve replacement in these generally critically ill patients is also widely appreciated. The main risks of thrombolytic treatment are the thromboembolic complications, which appear in from 4% to 13% of the patients, and bleeding, which occurs in from 1.4% to 5%.[3]

We have read with interest the excellent report written by Nguyen and collegues.[4] They have added an important case to the medical literature for the successful application of the thrombolytic protocol with recombinant tissue-type plasminogen activator (rt-PA), which has not been used previously in the management of PVT. It consisted in a continuous intravenous infusion of rt-PA at a rate of 1 mg/h together with the administration of heparin in a continuous intravenous infusion of $3\ U \cdot kg^{-1} \cdot h^{-1}$. The duration of treatment was 80 hours. At the end of the fibrinolytic infusion, the transprosthetic gradients had decreased from a peak and mean of 158 and 86 mm Hg to 48 and 25 mm Hg, respectively. Fluoroscopy confirmed normal motion of the prosthetic valve. The patient's symptoms resolved.

We would like to make some comments related to this therapeutic regimen. Treatment with rt-PA in PVT has not been widely used. It has been blamed for a major risk of embolism other than thrombolysis for its potential and velocity of the infusion. Shapira and collegues[5] proved the efficacy and safety of rt-PA, with the additional advantage that if the thrombolytic treatment fails, surgery can be used with less risk for its less lytic systemic effect.

The regimen of administration is not well defined. This protocol probably needs a longer course and lower dose to provide better thrombolytic efficacy with less risk of complications in hemodynamically stable patients, because they do not need a prompt thrombolytic effect. An accelerated protocol with rt-PA should be reserved for critically ill patients.

Until now, streptokinase is the most effective thrombolytic agent used, alone or as a part of a sequential fibrinolytic treatment in the PVT.

Despite the favorable evidence of thrombolytic therapy in the treatment of the PVT, more data should be gathered to obtain a general consensus of the ideal management of this complication.

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Optimizing selective cerebral perfusion in adult aortic arch repair: Clinical relevance of the laboratory model

To the Editor:

I read with great interest the excellent article by Halstead and colleagues[1] detailing in their porcine model of deep hypothermic circulatory arrest (DHCA) the neuroprotective effects of selective cerebral perfusion (SCP) via both carotid arteries at a mean of 50 mm Hg for a period of 90 minutes. In this laboratory model, the authors have clearly demonstrated the adverse cerebral effects associated with SCP at higher pressures and flow rates. The clinical relevance of this observation is illustrated in the study by Khaladji and colleagues,[2] in which they analyze outcomes after hypothermic circulatory arrest (71.1% hemiarch; 10.4% total arch) and bilateral cold selective SCP at a perfusion pressure of 40 to 60 mm Hg with flow rates of 400 to 650 mL/min.

However, Halstead and colleagues chose a long SCP time of 90 minutes, which is the time required for a total arch repair. In an earlier clinical study, these investigators[3] demonstrated their technique with a trifurcated graft with mean DHCA/SCP times of 31.1 $\pm$ 6.6 minutes and 65.3 $\pm$ 20.9 minutes, respectively, with SCP perfusion pressures of 50 to 70 mm Hg with flow rates of 800 to 1200 mL/min. Hence, this latest laboratory study is part of their ongoing quest to optimize their technique of total arch replacement with SCP, and it suggests a new range for bilateral SCP perfusion pressures and flow rate.

However, although this model is clinically relevant for hemiarch repairs,[2] how might it apply in the case of aortic arch repair with unilateral SCP?[4] Would lower SCP perfusion pressures be clinically superior, assuming a clinically competent circle of Willis? Or would the contralateral brain be at significant risk of ischemia, given the relevant incidence of clinical inadequacy in the circle of Willis for cerebral perfusion in DHCA with unilateral SCP?[5] Do the authors plan to evaluate unilateral SCP in their porcine DHCA model?

I congratulate the authors again on their important contribution. I look forward to their comments about these aspects of selective cerebral perfusion during adult aortic arch repair.

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